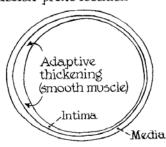
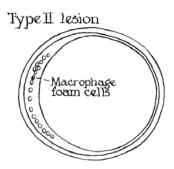
## 9. Atherosclerosis-Pathogenesis

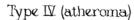
Coronary artery at lesion-prone location





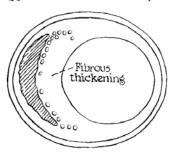
Type III (preatheroma)



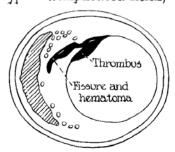




Type V (fibroatheroma)



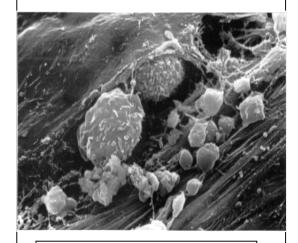
Type VI (complicated lesion)



Nomenclature and main histology	Sequences in progression	Main growth mechanism	Earliest onset	Clinical corre- lation
Type I (initial) lesion isolated macrophage foam cells	Ţ		from	
Type II (fatty streak) lesion mainly intracellular lipid accumulation	Ť	growth mainly by lipid accumu- lation	first decade	clinically silent
Type III (intermediate) lesion Type II changes & small extracellular lipid pools	(iii)		from third decade	
Type IV (atheroma) lesion Type II changes & core of extracellular lipid	Ţ			
Type V (fibroatheroma) lesion lipid core & fibrotic layer, or multiple lipid cores & fibrotic layers, or mainly calcific, or mainly fibrotic		accelerated smooth muscle and collagen increase	from fourth decade	clinically silent or overt
Type VI (complicated) lesion surface defect, hematoma-hemorrhage, thrombus	Ų (VI) —	thrombosis, hematoma		

## Coronary Atheroma-Infectious?

- Fluorescein labeled Antibodies to Chlamydia antigen in coronary plaque Organism in 79% of atheroma 4% normal vessels Muhlestein, JACC 1996
- Antibiotics reverse plaques in rabbits
  Muhlestein, Cir 1998
- Nanobacteria, Nanobacterium sanguineum,



**Monocytes under Endothelial** 

## Atheroma Ossification not Calcification (1863)

- Calcification in Atherosclerosis is similar if not identical to calcification seen in Ghon Complex of Tuberculosis.
- Initial calcification leads to formal lamellae bone with osteoclasts and osteoblasts, even marrow appears!
- Heavily Calcified Coronaries are due to dense chronic Osteoclastic remodeling.

Virchow, 1863